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Kidney Function Tests

As an introduction to assessment of tests of renal function, with emphasis on those procedures applicable clinically for evaluating renal function in patients, it is appropriate to summarize recent advances regarding the varied functions of the kidney.

Functions of the Kidney

The kidney is the organ on which depends the dynamic stability of the internal environment of the body and subsequent maintenance of a steady state. It is mainly responsible for regulation and maintenance of fluid, electrolyte, osmotic, and acid-base balances.

The kidney plays at least a fivefold role in maintaining the internal environment of the body in a steady state: (1) It eliminates water when excessive amounts tend to decrease the osmotic pressure of body fluids. (2) It excretes certain substances normally present in the blood when their concentration exceeds a certain threshold. (3) It normally conserves and selectively reabsorbs substances valuable to the body economy, such as glucose, proteins, amino acids, vitamins, and hormones as long as their concentration is commensurate with bodily demands and does not exceed the maximal capacity of the renal tubular mass. (4) It excretes substances that are useless to the body, no matter what their concentration in the blood may be and no matter whether they are end products of metabolism or are introduced into the body from exogenous sources. (5) It aids in maintenance of acid-base balance by formation and substitution of ammonia for inorganic base, excretion of monohydrogen or dihydrogen salts to eliminate or save base, excretion of variable amounts of inorganic base such as bicarbonate, and elimination of fixed acids.

In order for the kidneys to function normally with efficient excretion of endogenous and exogenous metabolic wastes, certain major physiologic conditions must be fulfilled. Factors contributing to normal renal function include: (1) total volume of circulating blood, (2) rate and quantity of blood passing through the kidney, (3) hydrostatic and colloid osmotic pressure of the blood, (4) intrarenal, luminal, and capsular pressures, and (5) morphologic status of the glomeruli and tubules. The kidneys are richly supplied with blood, the total flow to both kidneys being approximately 1300 ml per minute or practically a fourth of the total cardiac output.

The kidneys manufacture urine as a by-product of their major functions in regulation and maintenance of fluid, electrolyte, osmotic, and acid-base balances.

Functional Means of Renal Reserve

In addition to the amazing regenerative capacity of renal epithelium and the possibility of intermittent glomerular activity, the kidney has certain

functional means of reserve. It has been demonstrated that removal of 50 to 66% of renal tissue from the dog does not produce any detectable change in urinary volume or urinary nitrogen. The amount of functioning renal tissue must be reduced to about a third of normal before the values of various metabolites in the blood normally excreted by the kidney begin to increase. More than three-fourths of the renal substance must be destroyed before dogs die from renal insufficiency.

When parts of the renal tissue become functionless, the nephrons that remain healthy compensate for the destroyed nephrons by undergoing hypertrophy—work hypertrophy. Another means of renal reserve is an interplay of physiologic mechanisms that lead to increased filtration pressure and reduced reabsorption. High blood pressure in some types of renal disease increases filtration; the large volumes of dilute urine in some patients may compensate for inability of a diseased kidney to concentrate. A much larger volume of urine is eliminated to rid the body of the usual quantity of solids.

If the urine has a consistently fixed specific gravity near 1.010 when the intake of fluid is restricted, the inference is that the kidneys are unable to concentrate. The specific gravity of such urine—the same as that of the glomerular filtrate—indicates that the renal parenchyma is so severely damaged that it is not capable of doing osmotic work. Normal kidneys can concentrate urine to 1.035 or more, and can dilute to 1.000.

Renal Control of Sodium and Potassium

Sodium. Sodium and chloride are present in the glomerular filtrate in practically the same concentration that they are in plasma. Of the amount of sodium filtered in 24 hours, a total of about 1200 gm is reabsorbed, yet the average daily intake and excretion of sodium chloride is only 5 gm. Of the sodium that filters through the glomeruli, 85% is actively reabsorbed by the proximal tubules. The remaining is practically all reabsorbed actively by the distal tubules under the influence of aldosterone. Reduction in excretion of sodium chloride, with retention of sodium, can be explained only by diminution in the filtered load or by increased enzymatic activity induced by aldosterone.

Retention of salt in congestive cardiac failure is associated with changes in the renal filtration fraction. Renal blood flow decreases when the cardiac output becomes inadequate to meet the total metabolic requirements. This reduction in renal blood flow without much change in the glomerular filtration rate leads to an increased filtration fraction which brings about an abnormally high colloid osmotic pressure within the peritubular capillaries. This, in turn, promotes tubular reabsorption and leads to retention of salt and water. The accompanying increase in venous pressure in heart failure leads to transudation of fluids and stimulates the production of aldosterone. Thus, increased

filtration fraction, increased colloid osmotic pressure in the peritubular capillaries, elevated venous pressure, enhanced production of aldosterone and, later, reduced glomerular filtration, all exaggerate retention of salt and water in the terminal phases of congestive cardiac failure.

Potassium. Formerly it was considered that active reabsorption is the only tubular process involved in excretion of potassium. Recently, it has been shown that there is tubular excretion, probably by the distal tubules. Excretion of potassium by the tubules is related to the potassium content of the tubular cells rather than the concentration in plasma. It is not influenced by the filtered load or by urinary flow.

Function Tests Useful Clinically

Urea Clearance. Urea is the chief nitrogenous product of protein metabolism and is the chief nitrogenous constituent of the urine. Urea constitutes the largest quantity of the nonprotein nitrogenous compounds that accumulate in the blood. It is one of the most diffusible organic constituents and is found throughout body fluids. It is completely filtrable; during maximal diuresis of water, about 40% of the filtered urea is reabsorbed by passive diffusion through the various segments of the tubules. At a slower urinary flow, such as 1 ml per minute, more urea is reabsorbed resulting in a reduction of urea clearance.

Urea clearance should be measured when the urinary flow is either constant or decreasing because, during increasing diuresis, exaggerated and falsely high values may be obtained. Urea clearance is an expression of the combined excretory functions of filtration and reabsorption. Normally, it is 65% of the simultaneously determined inulin clearance, which indicates that 35% of the filtered urea is reabsorbed in its passage down the tubules.

Creatinine Clearance. Creatine is converted to creatinine but not vice versa; the only sources of urinary creatinine in a person consuming a creatinine free diet are muscle creatine and phosphocreatine. Of the substances ordinarily excreted by the kidney in man, creatinine is concentrated to the greatest extent. It is now known that, in addition to elimination by glomerular filtration, creatinine is also eliminated by tubular secretion. For a variety of reasons, creatinine clearance is a good practical clinical test of glomerular filtration rate.

Blood Urea and Creatinine. The amount of creatinine and urea in the plasma increases when the glomerular filtration rate decreases. However, this increase is rather insignificant until loss of renal function becomes severe. When the blood level of creatinine is more than 2 mg/100 ml and that of urea is more than 60 mg, a severe depression in the glomerular filtration rate is expected. The rate of increase in the amount of urea and creatinine in the blood is not so fast as the rate of reduction in the glomerular filtration rate during the initial days of acute renal failure. The concentration of creatinine

and plasma, being influenced much less than is urea by diet and other factors, permits a much more reliable assessment of the glomerular filtration rate and should be utilized more often for that purpose.

Concentration and Dilution Capacity. The concentrating ability of the kidney as obtained from measurement of the specific gravity is a good index of functional integrity. Specific gravity should not be determined in glassware washed with detergents because such agents decrease the surface tension and increase the specific gravity. Also, freshly voided urine should not be tested; urine should be tested at room temperature. For every 3 C increase above the standard temperature of the hydrometer, the measured specific gravity is 0.001 less than the true value.

With progressive kidney destruction, a point is finally reached at which all urine excreted contains a relatively constant amount of total solids. At that stage, the specific gravity becomes fixed at 1.010 which is that of the glomerular filtrate. After this stage, the test is no longer of aid in evaluating further impairment in renal function in the course of renal disease.

The ability of the kidneys to dilute is not so valuable as is their concentrating capacity in the evaluation of renal function because it is lost relatively late and reappears rather early.

Excretion of PSP. Phenolsulfonphthalein combines reversibly with plasma albumin. Of the total dye in the plasma, 94% is excreted by tubular activity, the remainder by filtration. The capacity of the kidney to excrete PSP is measured by determining the rate of excretion in a fixed period or the rate of its clearance from the blood. The normal value for clearance of PSP from the blood is about 400 ml per minute. The excretory capacity of the kidneys for PSP is affected by specific impairment of the tubules that excrete the dye and by a decrease in effective renal blood flow. It must be remembered that administration of 6 mg of PSP does not increase or maintain plasma levels that are high enough to reach the maximal tubular excretory capacity and, therefore, will not indicate early impairment of renal function except when fractional samples are taken during the first hour.

Battery of Renal Function Tests

To pinpoint accurately the diagnosis of renal disease, a battery of tests carefully done and evaluated, yields much more helpful information than does one test performed on a single occasion. Starting with the simplest procedure, and going on to other tests deemed advisable is the procedure of choice. (K. G. Wakim, Appraisal of Kidney Tests: J Urol, 84: 1-9, July 1960)

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Clinical Manifestations of Infectious Mononucleosis

The clinical picture of any disease emerges most clearly if one physician examines and interrogates every patient, records observations, and draws conclusions. Neither complete nor reliable information is acquired from retrospective review of clinical records of other—often less interested and experienced—observers.

Medical literature contains tables of symptoms, but few detailed reports, of the clinical aspects of infectious mononucleosis. The author, having noted discrepancies between his observations of patients with infectious mononucleosis and statements in textbooks, evaluates his own clinical experience with 200 consecutive cases, and presents a detailed description of mononucleosis as it evolves in hospitalized patients.

All patients had the characteristic blood smears and heterophil antibody reactions of mononucleosis. Therefore, clinical observations presented are considered to be unalloyed by inclusion of illnesses with misleading similarities with mononucleosis. Minimal hematologic requirements for diagnosis are: (1) lymphocytes must constitute over 50% of leukocytes; (2) "atypical" lymphocytes must be present; (3) both of these features must be present over a period of at least 10 days. The last requirement will eliminate some viral disease which may produce transient lymphocytosis with atypical lymphocytes. All patients had characteristic heterophil antibody reactions: sheep cell agglutination titer of at least 1:56 in unabsorbed serum; a titer of at least 1:28 after absorption of serum with guinea pig kidney; and no agglutination after beef cell antigen absorption.

Seemingly, it has never been pointed out that mononucleosis is unusual among acute illnesses in that it evolves relatively slowly. Most uncomplicated acute diseases produce characteristic symptoms and signs in 3 days or less after onset—not mononucleosis. Whenever blood counts are characteristic of acute microbial diseases, they too usually appear within the first few days. In mononucleosis, characteristic blood counts are often delayed until the second week.

Inasmuch as mononucleosis has not been transmitted experimentally, and because patients usually give no history of contacts with other patients, one can only speculate about the incubation period. Circumstantial evidence points to an incubation period of 33 to 49 days.

The clinical picture of mononucleosis becomes recognizable and characteristic as the days elapse. During the first 2 days, most patients experience only malaise. Fever, chilliness, and malaise increase and sore throat and headache usually develop by the fifth day of illness. Physical examination at this time usually reveals a fever around 100 to 101 F, slight posterior cervical and axillary lymph node enlargement, and pharyngeal inflammation. Although jaundice usually appears after patients have been ill more than 7 days, about 1 to 2% will seek initial medical attention because of jaundice.

On the basis of the clinical picture determined following recovery, three syndromes are proposed with the percentage of occurrence in the author's series: pharyngeal, 80%; typhoidal, 12%; icteric, 8%.

Subjective manifestations of mononucleosis are characteristic but have little diagnostic value. The symptoms were usually mild or moderate in the first week; patients experienced malaise but not prostration; fever was not extreme; patients often felt chilly but rarely experienced chills; headache was rarely severe; jaundice was never intense. Sore throat was often a presenting complaint but at times developed later. Not all patients with visible pharyngeal inflammation complained of sore throat—76% complained of moderately severe sore throat, 11% had severe pain interfering with swallowing.

Some symptoms rarely—or never—encountered by the author are stressed as being of considerable value in militating against the diagnosis of uncomplicated mononucleosis: chest pain, nasal discharge or congestion, paroxysmal or harassing cough, sputum, joint pains, painful or extremely tender lymph nodes, watery diarrhea, hematuria or dysuria, and severe abdominal pain. Some of the enumerated symptoms have been stated to occur in mononucleosis; in many instances, such diagnoses antedated use of the heterophil reaction; in other cases, the diagnosis was made regardless of the reaction or without using absorption techniques; in a few cases, two diseases probably coexisted.

Lymph node enlargement was invariably present at some time; usually detectable by the end of the first week of illness. Of diagnostic significance was enlargement of the posterior chains of cervical nodes, often palpable down to the clavicle. Enlargement of axillary and inguinal nodes was usually, but not invariably, present. There was no correlation between degree of lymph node enlargement and degree of pharyngitis or duration of illness. In relation to lymph nodes, local heat and redness, marked tenderness, fluctuation or suppuration, or unilateral involvement was never noted.

Pharyngeal inflammation varied in intensity; only 25% of cases were severe. Exudate, usually spotty, was observed in 22% of cases; hyperplasia of pharyngeal lymphoid tissue was almost always visible. Usually, pharyngitis increased in severity for several days, reached its peak within 5 days, remained unchanged for 2 to 3 days, and then rapidly subsided.

Edema of the eyelids was noted in 36%, usually occurred within the first 10 days of illness, and lasted only about 3 days.

Tha palatal enanthem is not considered specific for mononucleosis. Nevertheless, when all other characteristic clinical features of mononucleosis are present, the appearance of this sign will almost certainly confirm the diagnosis.

Jaundice was observed in 16 patients, appearing between the fourth and fourteenth day of illness. It was usually mild, never severe. In some instances, mononucleosis had been diagnosed or suspected; in others, "viral hepatitis" had been diagnosed.

Rash was considered to be of negative value in diagnosis of mononucleosis; it occurred in only 3% of cases. Because skin eruptions appear much more frequently in several other acute febrile diseases, it seems logical that their presence should cast doubt on the probability of mononucleosis. Recrudescences after 2 to 3 afebrile days occurred infrequently and lasted only 2 to 4 days; relapses after 5 or more fever-free days never occurred. "Chronic mononucleosis" is a misconception. From the medical literature, there is the impression that complications occur frequently. In the author's series, this was not the case. One patient's spleen ruptured spontaneously. Facial nerve paresis, mild meningitis, salivary gland inflammation, and complete atrioventricular heart block were each observed only once.

From personal observations, the author seeks to remove the adjective "protean" from its often repeated, but unjustified and incorrect, association with mononucleosis. Clinical manifestations were not manifold or bizarre, and the symptoms, although not all specific, were so uniform that interrogation alone helpfully guided thoughts to, or away from, mononucleosis. (COL R.J. Hoagland MC USA, The Clinical Manifestations of Infectious Mononucleosis - A Report of Two Hundred Cases: Amer J Med Sci, 240: 21-29, July 1960)

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Steroids in Management of Selected Infectious Diseases

A few years ago, it was pointed out that judicious use of adrenocorticotropin and corticosteroids frequently contributed to immediate improvement in the condition of patients seriously ill with infections and their complications. Additional evidence has been widely documented in the literature. That these steroids were potentially dangerous agents and should be administered cautiously was also emphasized. However, except in rare instances, accumulated clinical experience has not demonstrated serious side effects when the adrenocorticosteroids were used over a brief period of time in critically ill patients.

The author presents a summary of his experience during the years, 1950 - 1959. The basic philosophy underlying therapy was use of pharmacologic doses of adrenal steroids for a period of a few days in critically ill patients.

Steroid Therapy in Shock Due to Infection

Probably there is no area in which further information is more urgently needed than in the problem of patients with peripheral vascular failure due to infection. Recent observations have indicated that shock which results from

overwhelming infection is not due to hemorrhage and necrosis of the adrenals as previously supposed to occur in such conditions as the Waterhouse-Friderichsen syndrome.

Antibiotics, infusions, pressor agents, steroids, and oxygen were employed in most of the patients with severe bacterial infections. Pressor drugs were used with caution in dosage to maintain the blood pressure around 90 mm Hg, rather than higher, to prevent severe vasoconstriction, but at the same time to increase cardiac output and stabilize the blood pressure. Supplemental oxygen therapy appears to be advantageous because studies on endotoxin shock in the laboratory have shown the protective effect of oxygen.

Although the mortality rate (60%) in this group was high and evaluation of steroid therapy difficult, it was concluded that intensive therapy was useful. Large doses of IV steroid—300 to 500 mg of hydrocortisone or equivalent, every 8 to 12 hours for 2 to 3 days—may be employed in the early stages of treatment without harmful effects. Definitive therapy remains unsatisfactory, largely because the mechanisms involved in pathogenesis of peripheral vascular collapse are not understood.

Steroids in Undesirable Inflammatory Reactions

Inflammation is an essential factor in the defense mechanism against infections, but inflammatory reactions can be harmful to the host and often unnecessary. Inflammation appearing during the course of an infectious disease frequently represents a delayed type of acquired microbial hypersensitivity; it can be especially harmful when certain organs are involved, such as the heart or central nervous system. Certain inflammatory states occurring in the course of infections can and should be subdued promptly.

Infectious Mononucleosis. Studies on unselected patients with infectious mononucleosis revealed that steroid therapy did not alter the clinical course favorably enough to warrant use of these agents in this disease except when certain complications appeared, such as inflammation involving the central nervous system.

Mumps. Steroid therapy should be considered in selected patients with complications of mumps. Such therapy should not be used in routine orchitis, but only for individuals in a febrile and toxic state and with painful effusion into the scrotal sac. Encephalomeningitis and thrombopenic purpura may be other complications requiring use of steroids.

Tuberculosis. From accumulated experience with steroids as adjuvant therapy in treatment of tuberculosis, three significant features emerge: (1) Surprisingly few cases of latent human tuberculosis have been reactivated as a result of steroid therapy. (2) Dissemination of active tuberculosis, even without simultaneous use of antituberculosis drugs, rarely occurs. (3) There are now clear indications for use of steroids in management of selected cases of tuberculosis in conjunction with antituberculosis agents. Such cases would

be those with severe complications of the primary infection and/or concurrent severe conditions of other etiology.

Brucellosis. In many respects, pathogenesis of the illness caused by brucellae is similar to that of tuberculosis—toxicity and inflammation can be partly related to appearance of acquired hypersensitivity to the invading organism. Toxicity in patients seriously ill with acute brucellosis can be eliminated rapidly by use of steroids. The Expert Committee on Brucellosis of the World Health Organization now advises simultaneous use of steroids and antibiotics in selected patients with brucellosis.

Acute Rheumatic Fever. It is still not clear whether steroids contribute favorably to the ultimate outcome of rheumatic fever in most patients. It is generally agreed, however, that steroids are beneficial in selected patients during a critical phase of their acute illness when inflammation is such a prominent and debilitating feature. Steroids have proved most useful in patients with pancarditis or in those with pericardial effusion in whom salicylates and other similar agents have failed to result in improvement. An unfavorable aspect is that treatment sometimes has had to be given for prolonged periods of time because relapses have occurred when the dose of drugs was reduced or omitted entirely.

Hepatitis. Steroids have not been employed routinely by the author for acute hepatitis of viral origin. They have been reserved for critically ill patients who were anorectic and who had impending hepatic coma.

Trichinosis. This is a disease in which acquired hypersensitivity to the parasite undoubtedly contributes to illness. In the author's experience, seriously ill patients responded well to use of steroids.

Pneumococcal Meningitis. This form of suppurative meningitis still remains a serious disease with a significant mortality rate. Of two such patients seen by the author and treated with steroids, both died. However, steroids would seem to be logical supplemental therapy.

Miscellaneous Infections. Steroids might be indicated in selected cases of meningococcal meningitis with peripheral vascular failure or manifestations of purpura, varicella encephalitis, subacute bacterial endocarditis, idiopathic eosinophilic pneumonia, and typhoid fever.

Steroid Therapy for Drug Reactions

ACTH and adrenocortical steroids have been highly effective in management of selected patients with severe drug reactions. Observations of the author confirm earlier studies of others.

Preparation and Dose

For patients with infection and peripheral vascular collapse, best results may be expected from large doses of a parenterally administered

steroid that is readily metabolized. It is recommended that 500 - 1000 mg of hydrocortisone be administered intravenously over a 24-hour period in 2 or 3 divided doses. Usually, this intensive therapy can be discontinued within 4 to 5 days. At times, oral preparations can be employed after initial intravenous administration of hydrocortisone and may be indicated if treatment must be continued for several days. For this purpose, one of the analogues, such as prednisone, methylprednisolone or dexamethasone can be used in equivalent doses. When large doses of steroids are given for only a few days, it is not necessary to reduce the dose gradually and to administer ACTH. ACTH is not recommended either as the sole agent or as supplementary treatment during this intensive period of therapy.

In the group of patients suffering from undesirable effects of inflammation, large parenteral doses of steroid are not imperative. Additional stimulation of the adrenal cortex with ACTH may be sufficient. For the critically ill patient, hydrocortisone, 100 - 200 mg every 8 to 12 hours, is indicated. Improvement is usually apparent after 24 to 48 hours; then oral preparations of steroids can be employed in reduced amounts.

When intensive steroid therapy is carried out for an illness for which no specific chemotherapeutic agent is available, antibiotics are not usually employed prophylactically. Prophylactic antibiotic therapy is not recommended when steroid therapy is used in treatment of trichinosis. On the other hand, an antibiotic would be given to a patient receiving steroid who has had agranulocytosis induced by a drug.

Side Effect of Steroids

None of the patients treated by the author in this study revealed significant ill effects when the steroids were administered for a period of only a few days, even when large doses were used. Benefits have far outweighed possible hazards. (W. W. Spink, Adrenocortical Steroids in the Management of Selected Patients with Infectious Diseases: *Ann Int Med*, 53: 1-32, July 1960)

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Etiology of Pancreatitis

In any large hospital experience, the etiology of pancreatitis can be determined in two-thirds to three-fourths of the patients. The majority of patients have pancreatitis associated with either gallstones or alcoholism. That gallstone and alcoholic pancreatitis are distinct entities is apparent because of clinical course, incidence of complications, and response to surgery of each type. Clinical classification of pancreatitis as acute, relapsing, and chronic is of only moderate aid—all types may occur in these stages.

Gallstone Pancreatitis

Gallstone pancreatitis is recurring pancreatitis. Attacks of pancreatitis vary from acute pancreatic edema and hemorrhagic pancreatitis to pancreatic sequestration. Attacks of gallstone pancreatitis often are mild and subside rapidly; others may produce a lethal necrosis. According to the "common channel theory," reflux of bile into the pancreatic duct or reflux of pancreatic and biliary products into the common duct is indicated as causing pancreatitis. On the other hand, presence of pancreatic duct obstruction is also postulated as a cause of pancreatitis. Removal of common duct stones will stop attacks of abdominal pain and prevent further elevations of serum enzyme concentrations.

Pancreatic complications in patients with gallstone pancreatitis are uncommon unless associated with alcoholism. The course then follows that of alcoholic pancreatitis. Even though recurrences of pancreatitis are the rule with retained gallstones, the insult to the pancreas at each attack does not result characteristically in fibrous replacement of the pancreas to the degree seen in alcoholic pancreatitis. In contrast to the favorable response of gallstone pancreatitis to definitive operation, removal of the noncalculous gallbladder as therapy for pancreatitis is useless. In the authors' experience, there was 85% recurrence in this type of patient.

Alcoholic Pancreatitis

Alcoholic pancreatitis is a disease of young adults, more commonly men, although women are not immune. The patients generally are found to have begun an early heavy alcoholic intake, between 15 and 30 years of age, and to have continued to overindulge in the ensuing years. After about 8 to 10 years of alcoholism, attacks of abdominal pain begin and become progressively more frequent. About one-half of these patients are dead by the age of 45 from pancreatic complications, chronic hepatic disease, gastrointestinal hemorrhages, or accidents which these people are prone to encounter.

There is nothing characteristic about an acute attack of alcoholic pancreatitis except that it is also recurrent pancreatitis. The patient often complains of acute abdominal pain, vomiting is prominent, the liver may be palpable, bowel sounds may be diminished or absent, the serum amylase is usually elevated, and tenderness in the epigastrium and right upper quadrant may be found. The clinical picture may combine facets of pancreatitis, gastritis, and cirrhosis into one syndrome.

After many years of recurrent attacks of pancreatitis, chronic pain results. After a period of several years, at least one-fifth of these patients develop pancreatic exocrine and endocrine insufficiency. The course from this point is progressively downhill. The absence of gallstones is striking. Should stones be present and cholecystectomy be done, the course of pancreatitis follows that of the alcoholic variety—one of continued recurrence.

One of the clear characteristics of alcoholic pancreatitis is development in a significant number of patients of chronic pancreatic complications—weight loss, steatorrhea, diabetes, and pancreatic calcification. The authors consider pancreatic calcification to be almost *prima facie* evidence of alcoholic pancreatitis. Calcification usually begins in the head of the pancreas and gradually spreads to involve the entire organ. Many patients have diffuse calcinosis when first recognized. Once calcific pancreatitis occurs, diabetes develops in approximately one-third. Pseudocysts are frequently found which is not the case in gallstone pancreatitis.

One characteristic of the patient with alcoholic pancreatitis seen today is the history of several operative procedures having been performed in an effort to control pain. From published reports, the advisability of attempting indirect operative procedures in this type of pancreatitis may be seriously doubted. Whether extirpative surgery will prove to be a solution for alcoholic pancreatitis remains to be seen.

Other Types of Pancreatitis

Associated with Hyperparathyroidism. This is one of the rarer forms of pancreatitis. To date, only 13 such patients have been reported. Clinically, in most instances, the presenting complaints were those referable directly to hyperparathyroidism. The pancreas in about one-half of these patients had diffuse calcification, bespeaking metastatic calcification associated with hyperparathyroidism.

Associated with Familial Hyperlipemia. This rare form of pancreatitis has been observed in young patients with hyperlipemia. To date, only 12 patients have been reported in detail. Abdominal pain may have occurred several times before hyperlipemia is recognized. At times, hyperlipemia coincides with the acute attack of pancreatitis to subside as symptoms subside. Xanthomatosis is occasionally observed. Pathogenesis of this unusual disorder is not clear.

Mumps Pancreatitis. The few published reports containing objective information leave a reasonable doubt concerning existence of this form of pancreatitis as a definitive entity. There is abundant evidence that parotitis itself is associated with persistent hyperamylasemia with normal or clinically insignificant rises in serum lipase values. Objective diagnosis of mumps pancreatitis will await development of a more specific index of disordered pancreatic function.

Postoperative Pancreatitis. Acute pancreatitis complicating operations in the upper abdomen has long been recognized. Commonly, pancreatitis complicates gastric surgery. It is evidenced by four clinical syndromes either occurring independently or in association with other forms: (1) acute benign pancreatitis; (2) operative injuries to the pancreatic ducts; (3) pancreatitis associated with leakage from the gastrointestinal tract; and (4) early postoperative fulminating pancreatitis. Operations about the biliary tract have

been noted occasionally to cause acute pancreatitis. When this occurs following common duct exploration, it is a catastrophic and fatal complication. Mortality as high as 83% has been reported. The patient who survives the acute attack of postoperative pancreatitis rarely develops the syndrome of recurrent pancreatitis.

Idiopathic Pancreatitis. This designation is used when none of the other etiologic factors known to cause pancreatitis can be demonstrated. The importance of such a designation lies in the realization that there are other forms of this disease with etiologic factors yet to be determined. (J.M. Howard, E. W. Ehrlich, The Etiology of Pancreatitis: Ann Surg, 152: 135-146, July 1960)

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The Mallory-Weiss Syndrome

Massive upper gastrointestinal hemorrhage originating from lacerations of the cardio-esophageal region which is caused by repeated vomiting was first described by Mallory and Weiss in 1929. Many of the original, as well as the few subsequently reported, cases must be considered equivocal. Despite the obvious assumption that such a paucity of case reports would indicate that this is a very rare disease, it has been the author's contention that a lack of awareness of such an entity on the part of the clinician, surgeon, and pathologist has resulted in failure to recognize the disease.

The description of the lesions in the original reports as well as in subsequent cases, including two described by the author, are all in close agreement. The lacerations occur in the longitudinal axis and are arranged in a radial manner anywhere about the cardio-esophageal junction. They may appear in the esophagus or stomach alone, or extend into both. Frequently, they are multiple; up to five in a single case have been reported. The lacerations may extend completely through the muscularis mucosae. In nearly every instance, the appearance of the laceration, either grossly or microscopically, is that of a fairly fresh tear, although one or two cases have been reported with further complications of a chronic nature, such as eventual delayed perforation.

There has been considerable speculation about the exact etiology of these lacerations, but no better explanation has been offered than the original concept that forces exerted at the time of vomiting cause the tears. Mallory and Weiss believed that the usual highly coordinated reflexes occurring in the act of vomiting become disturbed; the esophagus and diaphragm then fail to relax, particularly after repeated attacks of vomiting and retching. As a result, the gastric contents are thrust with great force against the cardiac opening.

Certain nutritional and other types of disease might well contribute to occurrence of these tears, as one would certainly anticipate a more fragile

mucosa in the presence of severe nutritional imbalance or marked alcoholic or atrophic gastritis. However, despite the more frequent than average appearance of these factors in such cases, the one common denominator always present has been vomiting.

To increase accuracy in diagnosing the syndrome clinically or surgically, the possibility of such a lesion being present in all cases of unexplained hemorrhage, especially massive upper gastrointestinal hemorrhage, must be kept in mind. If the history is clear, with the story of vomiting normal stomach contents on one or more occasions prior to massive hematemesis or hematochezia, then one must be highly suspicious of the presence of such a lesion even in the face of a history suggestive of ulcers or cirrhosis. Roentgenograms, as would be anticipated, have been of no benefit whatsoever in diagnosing this lesion.

In view of the hesitancy of many endoscopists to introduce an esophagoscope, the final diagnosis in most cases will probably depend on the findings at surgical exploration.

Treatment should start, as in most cases of extensive upper gastrointestinal hemorrhage, with at least a brief period of conservative management and careful observation. Use of the Sengstaken tube has been tried, but it is suggested that the pressure applied by the balloon is insufficient to control the arterial type of bleeding which is most frequently encountered with this syndrome.

When these measures have failed, prompt exploration is indicated. At the time of laparotomy, if there is no obvious cause of the bleeding, it is recommended that a generous anterior gastrotomy incision be made to enable thorough inspection and palpation of all portions of the stomach and down into the duodenum. If bleeding is extensive, making localization of the source difficult, it has been suggested that the mid-portion of the stomach be packed so that it can be determined whether the bleeding is coming from above or below that level. Once the lesion is visualized, simple transfixion of bleeding points with catgut sutures and closure of the laceration with the same type of material has been the treatment used by all of those reporting.

The importance of recognizing and promptly handling the situation at surgery is very much in evidence in reviewing the cases in the literature because the results of doing too little or too much at the time of exploration is vital to the patient's welfare. (S.N. Etheredge, The Mallory-Weiss Syndrome: Amer J Surg, 100: 200-205, August 1960)

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If I were to prescribe one process in the training of men which I consider fundamental to success of any kind, it would be thoroughgoing training in the habit of accurate observation. It is a habit which every one of us ought to be seeking ever more to perfect.

—Eugene G. Grace

Management of Injuries to Major Veins

Although considerable attention has been paid to vascular trauma, most of the emphasis has been on arterial, rather than venous, injuries. The authors present 52 injuries to major veins occurring in a series of 228 patients with vascular injuries.

The vein most frequently injured (9 times) was the superficial femoral. The internal jugular and the inferior vena cava were each involved 8 times. The brachial was injured in 7 cases, but the axillary only once. The popliteal was involved in 4 instances. Other major veins injured sporadically were the external iliac, innominate, hypogastric, common iliac, common femoral, external jugular, superior vena cava, and portal.

The mortality rate of nearly 20% in this series underscores the fact that major venous injuries cannot be dismissed lightly. While the high mortality rate in the reported cases was due in 6 instances to associated injuries to the head, chest, abdomen, and arteries, 4 of the 10 deaths were due directly to the venous injury. The over-all mortality rate compares closely with the mortality rate in vascular injuries of all types. In the present series, 4 of the 10 patients whose deaths were related to the vascular injury were not operated upon; one patient's injury to the inferior vena cava was not repaired at operation. It is possible that a more aggressive surgical attitude might have prevented these deaths.

There is a tendency to give patients who are in shock large quantities of blood before taking them to the operating room. As in other instances of hemorrhage, the best way to combat the shock is to stop the bleeding. This may require an aggressive approach to the major vessels of the neck, chest, and abdomen, often under less than ideal conditions, and frequently in the late hours of the night. It takes the greatest courage to desist from operating and the finest judgment to recognize the patient who will survive without operation.

Lateral suture repair should be done whenever possible. In the authors' experience, it was successful in every case; all patients who survived had good results. Ligation is usually satisfactory, but should be done only if lateral suture repair is not feasible. There has been a low incidence of thrombophlebitis after ligation. Heparin does not appear to be of value and has some obvious undesirable effects in massively traumatized tissue. Vigorous postoperative care of the patient who requires ligation of the inferior vena cava will prevent undesirable sequelae of this procedure.

Failure to accomplish ligation of major veins which have been injured produces poor results. In this series, there were 8 arteriovenous fistulas due to the fact that surgical exploration was not attempted. Five other patients died when injuries to major veins were not controlled. Only 2 patients who did not have operative control of a major venous injury recovered without sequelae.

Anastomosis of avulsed veins was not attempted in this series. It is doubtful that it would be successful because of the low pressure in the venous system. For this same reason, homografts and vein grafts or prosthetic grafts have not been used.

Eight injuries to the vena cava were of special interest. Five of the patients survived. Repair of the vena cava with lateral sutures is preferred to ligation. (M. R. Gaspar, R. L. Treiman, The Management of Injuries to Major Veins: Amer J Surg, 100: 171-175, August 1960)

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IN MEMORIAM

Lund, John A. CAPT MC USN (Ret) Anaheim, Calif.	19 May
Askin, Robert M. CAPT DC USN (Ret) San Francisco, Calif.	7 July
Dungan, William E. WO1 USN (Ret) U.S. Naval Hospital, Portsmouth, Va.	8 July
Wilson, Edward W. LTJG MC USN (Ret) Oakland, Calif.	21 July
Brown, James L. CDR DC USN (Ret) U.S. Naval Hospital, San Diego, Calif.	21 July
Ferguson, Russell S. CAPT MC USN (Ret) Ann Arbor, Mich.	22 July
Davis, Edgar F. LT MC USNR Jacksonville, Fla.	23 July
McKerley, Lowell H. LT MSC USN (Ret) Washington, D. C.	24 July
Lehman, Robert C. CAPT MC USN Ryukyu Army Hospital, Okinawa	25 July
Rossina, Wesley R. CAPT DC USNR Arlington, Va.	30 July
Ballard, Miriam F. LT NC USN (Ret) U.S. Naval Hospital, Bethesda, Md.	22 August

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Cooperative Program of NSHA and The George Washington University

RADM B. W. Hogan, Surgeon General of the Navy, and VADM O. S. Colclough USN (Ret), Acting President, The George Washington University have approved a cooperative academic program between the

Naval School of Hospital Administration, National Naval Medical Center, Bethesda, Md., and The George Washington University. By this arrangement, successful completion of courses at NSHA will be the basis for college credit for those officer students who meet regular admission requirements of the University. Credits earned during the 10-month course will be considerably more than normally earned in a typical college year and may be applied to an Associate in Arts or Bachelor in Arts degree. The cooperative program will begin with the class convening 22 August 1960. MSC officers will continue to be ordered to NSHA in accordance with the policy set forth in enclosure (1) of BuMedInst 1520. 12A.

To effectuate this program, an off-campus center of the College of General Studies of the University will be established at NSHA. Of the total credit courses, nearly one-third will be taught by university faculty members. The remainder will be taught by academically qualified MSC officers attached to NSHA who will be accorded appropriate university faculty status.

The Naval School of Hospital Administration had a modest beginning in 1942 as a school created to satisfy a heavy wartime demand for specialists in various aspects of hospital and medical administration. Emphasis was laid on property and accounting, personnel, and commissary management. At that time, it was a training department of the U.S. Naval Hospital of the National Naval Medical Center. Through the years, the scope of the curriculum has broadened and course content has been revised to meet changing requirements. In 1945, the Secretary of the Navy designated the school as the Naval School of Hospital Administration which is now under the command of a Medical Service Corps officer. The school continues to function as a command unit of the National Naval Medical Center under the management and technical control of the Bureau of Medicine and Surgery. CDR C.F. Johnson MSC USN is the present Commanding Officer.

The Surgeon General and the Acting President of the University have expressed confidence that this program will result in mutual benefits and fruitful relationships in the academic endeavors of the two schools.

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BUMED INSTRUCTION 6260. 10A

12 August 1960

Subj: Eye examinations of certain designated personnel assigned duty involving eye exposure to ionizing and nonionizing radiation

This directive provides for conducting eye examinations on designated persons, both military and civilian, in the Naval Establishment for the purpose of screening, detecting, and recording the earliest signs of lenticular opacities which may be due to exposure to either ionizing or nonionizing radiation, and recording preexisting or congenital lesions.

BUMED NOTICE 6230

11 August 1960

Subj: Influenza vaccination program for 1 October 1960 to 31 July 1961

This directive provides information concerning the utilization of polyvalent influenza virus vaccine by military activities during 1960-1961. During October 1960 polyvalent influenza virus vaccine shall be administered to all naval personnel on active duty—with certain exceptions. Immunization of dependents is desirable, particularly school-age children who are more susceptible than adults and are often the source of infection of their parents.

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Association of Military Surgeons Convention

On 30 October 1960, more than 2000 American and international physicians, dentists, veterinarians, nurses, and medical specialist delegates will begin registering for the 67th Annual Convention of the Association of Military Surgeons to be held at the Mayflower Hotel, Washington, D. C.

The theme of the 3-day convention is The Military Role in Medical Progress. RADM Richard A. Kern MC USNR (Ret), President of the Association, will open the meeting on 31 October. RADM Curtiss W. Schantz DC USN, Assistant Chief of the Bureau of Medicine and Surgery (Dentistry) is the General Chairman; CAPT Clifford P. Phoebus MC USN, Director, Astronautical Division of the Bureau, is Chairman of the Scientific Program Committee.

Among the series of papers with a wide range of interest are: Lessons Taught the United States by Mobilization for War, Defensive Aspects of Biological Warfare, Medical Problems Encountered in Antarctica, Recent Developments in the Treatment of Hypertension, Use of Frozen Tailored Blood, Aeromedical Support of the X-15 Program, Achievements in Thoracic Surgery, and Current Concepts of Management of Snakebite. Cholera, and Man and the Radiation Hazard will be covered by two important panel discussions. The William C. Porter Lecture, sponsored by Smith, Kline, and French Laboratories in honor of a pioneer in military psychiatry will be presented by Dr. Manfred S. Buttmacher, Chief Medical Officer of the Medical Service of the Supreme Bench of Baltimore.

During the sessions, there will be special section meetings with panel discussions for dentists, nurses, medical specialists, and veterinarians. Films on medical and scientific subjects will be shown continuously throughout the meeting.

Reserve officers attending the meeting may register for credit points. There will be no registration fee. Non-members are invited.

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Change in Promotion Plan for Warrant Officers

In his most recent letter to all Medical Service Corps and Medical Service Warrant Officers, the Chief of the Medical Service Corps provided information on the FY 1961 promotion plan. Included, as a matter of interest to warrant officers, was the plan for a "one-time selection" to temporary officer grades, previously announced in the Navy Times, 25 May and 11 June 1960.

The Bureau of Personnel now advises that a significant change is being made in provisions for eligibility. It is now planned to consider all warrant officers, regardless of age or length of service. This is being announced in the Navy Times with such other details as appropriate. Since the special board will convene in September, time will not permit issuance of an official notice.

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From the Note Book

Medical Training in the Navy. Figures have just been released which show that 174 Navy Interns completed their training during the past year, while 175 started internships on 1 July 1960. Medical officers selected to start residency training totaled 155. (TIO, BuMed)

American Board Allows Credit for Active Duty. The new Bulletin of the American Board of Obstetrics and Gynecology (1960) contains modifications which enable physicians in this specialty to apply active duty time in the Armed Forces towards requirements of the Board. Henceforth, actual practice of obstetrics and gynecology in the military service can be counted towards the 2 years of required postresidency experience.

Papers Invited for ACP Meeting. The 42nd Annual Session of the American College of Physicians will be held at Miami Beach, Fla., 8 - 12 May 1961. The Committee on Program is inviting original contributions for the scientific program, including: (1) Basic Science related to Internal Medicine; (2) Reports of Clinical Investigation; (3) New and Original Observations in Clinical Medicine. The meeting offers an excellent opportunity for presentation of new work to a large and representative group. Participants need not be members of the College. Informative abstracts of papers, suitable for publication in the program, if accepted, should be approximately 250 words in length. An original and 4 copies should be forwarded by 15 October to: Edward C. Rosenow Jr, M.D., Executive Director, American College of Physicians, 4200 Pine St., Philadelphia 4, Pa.

Aviation Examining Facility Relocated. The Aviation Examining Facility, formerly located in the Medical Department, U.S. Naval Air Station, Pensacola, Fla., was physically moved to the Naval School of Aviation Medicine in July 1960. In the future, all inquiries concerning flight physical examinations should be directed to the Commanding Officer, U.S. Naval School of Aviation Medicine, U.S. Naval Aviation Medical Center-54, Pensacola, Fla., rather than to the Commanding Officer, Naval Air Station.

Urological Seminar. The 8th annual meeting of the James C. Kimbrough Urological Seminar will be held at Walter Reed General Hospital, Washington, D. C., 3 - 5 November 1960. Although sponsored by the Army, all United States governmental urologists, military and civilian, are invited to attend. The contemplated program should prove appealing to urologists of all levels of training and experience. Prominent civilian participants will include John Kingsley Lattimer, William Wallace Scott, Richard Chute, Hugh J. Hewett, and William H. Boyce. Several panel discussions of important and controversial subjects are planned, and the popular pyelographic conference and case report session will be included. (WRAH, WRAMC)

Specialty Group Committee for Military Fellowships. A Committee on Fellowship of Armed Services Personnel has been appointed by the American College of Obstetrics and Gynecologists. This committee will process applications for fellowship and junior fellowship from physicians who are in the military service. CAPT S. L. Arje MC USN, Bureau of Medicine and Surgery, has been appointed to the committee to represent the Navy Medical Department.

American Psychology Association. The Surgeon General has announced that for the first time plans have been made to present both the Navy's clinical psychology and aviation experimental psychology programs simultaneously at a meeting of the American Psychology Association. This is expected to enhance the professional stature of psychologists in uniform. (TIO, BuMed)

CAPT Anderson Receives Decoration. The Rhode Island group of the U.S. Volunteer Life Saving Corps recently presented CAPT Edward A. Anderson MC USN with the Gold Medal of Honor—the highest decoration of the Corps—for his efforts to help stem the spread of a dangerous poliomyelitis epidemic in the state. Working with five Navy medical technicians during off-duty hours and using the pistol shaped, rapid firing vaccine injection guns which deliver 1200 painless inoculations an hour, Dr. Anderson and his team have inoculated more than 100,000 residents of Rhode Island with Salk anti-polio vaccine to date. The vaccine is provided by the State Department of Health and repacked at the Naval Air Station hospital for use in the "hypo-spray" guns. The team works on a seven-days-a-week basis, answering requests for "shooting session" from Rhode Island communities. (TIO, BuMed)

Foreign Officers Attend Courses at NMS. Some 20 foreign armed forces medical officers are attending courses in Naval Medical Management and Naval Preventive Medicine at the Naval Medical School, National Naval Medical Center, Bethesda, Md. Conducted concurrently for a period of 10 weeks, these courses are designed to prepare specially selected foreign officers of friendly countries for higher command responsibilities in their own navies, and to familiarize them with U. S. Navy methods, practices, and doctrines. (PIO, NNMC)

Navy Surgeons at Heart Surgery Conference. Three Navy Medical officers have been assigned to attend the Conference on Prosthetic Valves for Heart Surgery, sponsored by the Surgery Study Section, Division of Research Grants, National Institutes of Health, in Chicago, 9 - 10 September 1960. Major objectives to be covered include development, technical and clinical problems involved, results of past studies and experience, and areas in which further research is needed. Those attending are: CAPT R. B. Brown, Commanding Officer, U. S. Naval Hospital, Bethesda, Md.; CAPT J. J. Timmes, Chief of Surgery, U. S. Naval Hospital, St. Albans, N. Y.; CAPT R. L. May, Head, Surgery Branch, Professional Division, BuMed. (TIO, BuMed)

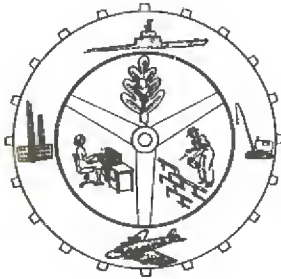
Poliomyelitis Incidence (Prepared by the National Office of Vital Statistics, and appearing in Morbidity and Mortality, Weekly Report of the Public Health Service, U. S. Department of Health, Education, and Welfare, 5 August 1960.)

	<u>1960</u>	<u>1959</u>	<u>1958</u>	<u>1957</u>
Week ending 30 July	108	307	159	297
paralytic	78	191	69	70
Cumulative Total (paralytic)	601	1338	579	885

Portacaval Shunt in Patients with Cirrhosis and Ascites. Considering the theory that increased venous pressure within the liver is responsible for, or contributes to, formation of ascites, the authors performed portacaval shunt procedures on 15 patients with medically irreversible ascites due to cirrhosis of the liver. Nine patients were considered to have experienced successfully cured ascites—all 9 having undergone end-to-side shunts. (H. Barker, K. Reemtsma, Surgery, July 1960)

Role of Liver in Ascites. In experimental animals, by moving the entire liver into the chest, the authors made observations that suggest that the source of experimental ascitic fluid is extruded hepatic lymph. (R. Aiello, et al, Surg Gynec Obstet, July 1960)

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OCCUPATIONAL MEDICINE

Treatment of Accidental Wounds

Man's environment is essentially a hostile one and many dangers threaten to lacerate or cause bursting wounds in the skin which envelopes him. Fortunately, the skin is a wonderful organ, and it is probably taken more for granted than any other organ we possess! Given the proper assistance and management, it has amazing abilities to repair and reconstitute itself. This clearly and unmistakably delineates the role of the physician who treats an accidental wound. His main objective must be to aid and promote healing.

Obviously, accidental wounds vary widely in type, location, and severity. However, it is possible to set down general principles of wound care and to offer sound advice concerning the technicalities of anesthetizing, suturing, and dressing.

Any discussion of accidental wounds must be prefaced by the repeated warning to evaluate the entire patient. Too often, attention is centered on the obvious exposed wound while other more serious wounds or injuries remain undetected and untreated. This is particularly true of extensive wounds about the head and face. Establishment of an airway and control of bleeding and shock always take precedence over any reconstructive procedures. After these immediate problems have been evaluated and controlled, the extent of other injuries—particularly head injuries—can be ascertained and intelligent decisions can be made concerning transfer of the patient to a suitable center for reconstructive surgery.

The Time Factor in Wound Care

Older surgical literature is replete with discussions of the so-called golden period—those first 6 to 8 hours after injury when the wound is considered to be contaminated but not infected and can safely be closed primarily. The advent of antibiotics has definitely changed this picture. If antibiotics are administered and the other principles of wound care are rigidly followed, primary closure can usually be carried out 14 to 16 hours after injury without the sequela of infection. This extension of time is particularly important in cases of facial lacerations in which primary closure offers the best chance

for a cosmetically acceptable scar. The excellent circulation of blood in the facial skin will reduce the odds against infections in such cases. However, facial wounds must be watched very carefully in the postoperative period. If suppuration develops, some or all of the sutures may have to be removed to establish drainage. In less cosmetically important areas, such as the trunk or extremities, the more conservative course of leaving the wound open for drainage and of simply covering it or packing it open with an ointment gauze may be the best choice. In 24 to 36 hours, such a wound could be sutured as a delayed or secondary closure with less chance of infection.

Anesthesia

The choice of anesthesia is usually apparent. If one is dealing with a single laceration or several small lacerations in an adult or manageable child, a local anesthetic is indicated. A solution of 1% lidocaine hydrochloride with epinephrine is satisfactory. When nerve blocks are performed on the digital nerves, the epinephrine should be omitted. The anesthetic agent can be injected within the wound if there is no gross contamination. Although this is an easier route and seems to produce less pain and discomfort for the patient, a field block done about the wound is preferred, because there is less distortion of tissue planes and less chance of leaving unanesthetized areas. The pain of injection can be reduced greatly if the solution is introduced slowly. Patients with extensive lacerations requiring several hours of surgery need a general anesthetic. If the lacerations are about the face, endotracheal intubation is necessary. This procedure enables the anesthetist to maintain both an adequate airway and full control of respirations, and it insures against the aspiration of blood and secretions. From the surgeon's standpoint, endotracheal anesthesia also has the advantage of getting the anesthetist out of the surgical field.

Local Care of the Wound

Local care of the wound is of utmost importance. To a large degree, it determines what the final scar will be—and there will be a scar, for scar tissue is the normal end product of wound healing. It is erroneous for a physician who is repairing lacerations—particularly those about the face—to assume that plastic revision will be necessary and inevitable in the future. An old axiom of surgery is that the surgeon who operates first has the best chance for a successful result. In other words, initial repair should be definitive; meticulous attention should be given to procedures which will promote good wound healing. These are: (1) proper cleansing of the wound, including removal of foreign bodies; (2) intelligent debridement with removal of nonviable tissue; (3) absolute hemostasis; (4) wound closure without tension

or dead space, using fine suture materials; and (5) immobilization with a properly applied pressure dressing where indicated and practical.

Cleansing. In larger wounds, the first step in cleansing is thorough irrigation of the wound with a running stream of normal saline solution. This mechanically flushes out clots, loose tissue, and small foreign bodies. The wound is then protected with sterile gauze while the surrounding skin is washed with an antiseptic detergent followed by a rinse of aqueous benzylkonium chloride. Routine use of strong antiseptics in the wound itself is to be condemned because they destroy not only bacteria, but tissue cells as well.

Debridement. Wound debridement should always be performed intelligently. Preferably, wound edges are excised cleanly and all nonviable tissue removed. If wound excision is not possible, as in cases in which vital facial structures are involved, the wound edges should at least be squared off so that they are straight and perpendicular. Such edges are much easier to suture, and produce more cosmetically acceptable scars. Bone fragments of any size are removed only if they have been stripped of periosteum and are lying free in the wound. A meticulous search should always be made for retained foreign bodies because in puncture wounds and flap wounds it is easy to miss small fragments of glass, metal, or wood. In particular, road dirt should be removed from wounds to prevent the objectionable pigmented scar. Larger particles can be picked out with the sharp point of a No. 11 scalpel blade. Finer particles ground into the dermis generally require judicious use of a scrub brush.

Hemostasis. Hematoma, one of the most notorious mechanical inhibitors of sound wound healing, can be prevented by careful hemostasis in which all bleeding vessels are tied with fine ligatures. No. 4-0 or 5-0 plain catgut is preferable, although nonabsorbable ligatures may be used. Particularly in facial wounds, hemostasis should be so complete that drains are unnecessary.

Wound Closure. The wound should be closed without tension on the tissues and without creating dead space. Tension on tissues leads to embarrassment or stoppage of the blood supply which culminates in necrosis and wound breakdown. Dead space will always fill with blood and tissue fluids, producing a mechanical barrier to wound healing and furnishing an excellent culture medium for infection. To attain closure without tension, the skin edges usually are undermined in all directions as far as the transverse dimension of the wound. The closure itself is accomplished by suturing the wound anatomically in layers, using fine instruments and fine suture materials. Gentle handling of tissues is emphasized because surgical trauma at the hands of the careless or hurried surgeon can raise more havoc with the wound than the original trauma. Fine instruments are essential to atraumatic handling of skin edges and should include a fine, single-tooth forceps; small mosquito hemostats; small curved sharp scissors; fine skin hooks; and a fine needle holder for instrument tying of sutures.

In layer closure, subcutaneous tissues usually are approximated with No. 4-0 plain catgut. The same kind of material is then used as a subcuticular suture to bring the wound margins together. This stitch is placed so that it grasps a good portion of the base of the dermis on either side of the wound, but is tied so that the knot is inverted. These subcuticular sutures really constitute the main strength of the sutured wound. When they have been placed correctly, the skin margins are carefully coapted with fine atraumatic No. 5-0 or 6-0 nylon or silk sutures. Since the surgeon does not rely on these fine-caliber skin sutures to hold the entire wound together, they can be more loosely tied and removed earlier, and thus are least likely to produce unsightly stitch marks. In actual practice, it must be admitted that many times poor end results are caused more by the way the suture is placed than by the caliber or type of suture material used. Precise apposition is essential to avoid uneven scars. The skin sutures usually enter at a distance from the edge of the wound equal to the skin thickness and are about 3 to 5 mm apart. It is desirable to have slight eversion of the skin edges to allow for scar contraction which inevitably follows in any healing wound. If the edges persist in being inverted, vertical mattress sutures are helpful.

Immobilization. After suturing is completed, the suture line is covered with a single layer of ointment gauze or one of the nonadhering dressings, such as rayon silk. Wherever possible, a pressure dressing of conforming gauze and elastic bandages should be carefully applied and maintained for 3 to 4 days. On the extremities, it is frequently possible to leave these dressings undisturbed until it is time to remove the skin sutures. Properly applied pressure dressings provide a degree of immobilization which aids wound healing and contributes to the patient's comfort. In addition, they also tend to prevent formation of hematomas, lymphedema, and venous congestion. In areas where pressure dressings are impractical, such as the labial and nasal areas, it is best to leave the area uncovered and to care for the open suture line. This includes cleansing the suture line with hydrogen peroxide applicators and applying an ointment, which may be simple sterile petroleum jelly or a topical antibiotic.

In administering tetanus prophylaxis and postoperative antibiotic therapy, each case must be individually considered. Patients who have incurred heavily contaminated crushing wounds on the highway require both types of therapy, while patients with relatively clean small incised wounds incurred in the home probably require neither. The bases between these two extremes pose problems and call for the clinical judgment of the responsible physician.

A question that is frequently asked in the postoperative period is: How long should the skin sutures remain in the wound? If large-caliber material has been used and the individual sutures have been tightly tied, the sutures should be removed in 2 to 3 days to avoid stitch marks. If finer caliber material, such as No. 5-0 or 6-0 nylon or silk has been used and the edges of

the wound have been gently approximated, the sutures can be left 4 to 6 days. In either case, it is wise to support the wound for an additional 4 to 7 days using nonflexible collodion applied over fine mesh gauze. This helps to minimize spreading of the fresh scar.

Special Types of Wounds

Additional suggestions for care of several special types of wounds may be helpful. At first glance, the more severe facial lacerations sometimes present difficulties in establishing points of reference. The eyebrows (which should never be shaved), the eyelid margins, the nasal alar margins, and the vermilion border of the lips can serve as guides. A few temporary tacking sutures using these guides are sometimes invaluable for preliminary orientation. At times, in severe facial wounds, there is appreciable tissue loss so that simple suturing will not suffice. If there is extensive loss of tissue about the mouth, or if there are penetrating wounds into the sinus cavities so that secondary reconstructive procedures will be necessary, it is important to suture the mucous membrane to the skin in order to control infection and minimize scar contracture. In other extensive skin losses, the choice might be between a free skin graft or a rotated flap. These are difficult and demanding cases, and should be done by surgeons experienced in reconstructive surgery if the best functional and cosmetic results are to be obtained.

In some wounds it is necessary to extend the laceration in the skin in order to visualize and repair some deeper structures. In such cases, the extensions should, whenever possible, be placed along the natural tension or wrinkle lines of the skin—the so-called Langer lines. To extend across these lines instead of along them is to encourage hypertrophic scar formation and scar contractures.

Every surgeon closing a skin wound hopes to obtain a fine hairline scar which will be cosmetically acceptable to him and to the patient. However, there are many variables in wound healing which are still not clearly defined or, if defined, are very difficult to control. This means that there are definite limitations to what can be done by any surgeon—including the plastic surgeon—in treating cutaneous wounds. Certainly, no surgeon should be so brash as to guarantee a good result nor should he lead the patient and his family to anticipate that the scar will be completely "invisible" or eventually will disappear altogether. Remember, these persons are called on to adjust psychologically to the presence of what is at best an unwanted scar no matter how excellent the result. This adjustment is made more difficult, or even impossible, if they have not been adequately prepared and advised.

(L. H. Backus, C. A. DeFelice, *Treatment of Accidental Wounds: Postgrad Med*, 27: 209-213, February 1960)

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Iron in Experimental Silicosis

In 1932, Kettle reported that when quartz particles are coated with iron their fibrogenic action is inhibited. Although initially unsuccessful, Gardner was able to confirm this inhibitory action in 1938. More recently, Vorwald also described the inhibitory action of iron on experimental silicosis in guinea pigs. Finely divided metallic aluminum powder (according to Denney et al.), and aluminum hydroxide (according to Gardner), also inhibit experimental silicosis. Gardner concluded that no inhibition could occur unless the inhibiting substance was within the same macrophage as the quartz particle it inhibited.

It is the purpose of this paper to describe the topographic relationship of iron and silica in the lungs of rats and guinea pigs in which experimental silicosis had been inhibited by iron, and to discuss certain findings which appear to be at variance with the above mentioned conclusion of Gardner concerning this inhibition.

Method

The silica was ground flint which was reduced to a size approximating 3 micra in a micronizer. The ground flint was suspended in water and under a pressure of 40 p. s. i. was forced through a fine nozzle of an atomizer at near-sonic velocity against a hardened tool-steel baffle, causing the particles to be broken into smaller fractions. It was apparently during this treatment that the silica became contaminated with iron. The vapor and suspended dust were passed next through an impinger which removed most of the coarser particles. The outflow from the impinger passed through an inhalation chamber and from there through electrostatic precipitators. The silica from the precipitators was used for intratracheal injections.

By x-ray diffraction, the silica dust consisted of $\pm 6\%$ alpha quartz and the rest consisted of micaceous material. No iron was found. By chemical analysis, however, 8 to 9% of the material was found to be iron. Failure to detect the iron by x-ray diffraction must be attributed to its noncrystalline state.

By electron microscopy, the average particle size of the silica was found to be 0.1 micron.

For control, the silica was treated with concentrated hydrochloric acid and washed with water until the silica and the wash water failed to react with acidified potassium ferrocyanide.

Both types of silica were suspended in water so that 1 ml contained 25 mg dust. One group of rats was injected intratracheally with 1 ml of the iron-contaminated quartz and another group of rats was similarly injected with the iron-free quartz. Some animals died immediately or within 24 hours after the injection. Others died at various times from intercurrent disease.

Pairs of animals were killed for sampling at various intervals up to 16 months following injection.

Guinea pigs were exposed to the iron-contaminated quartz dust in an inhalation chamber for 6 hours per day, 5 days per week, for one year. The dust concentration averaged 44 mg per cubic meter. Following the dust exposure, these animals were pastured for another year. At various intervals, animals were killed for sampling.

The lungs of all animals, whether dead of intercurrent disease or killed for sampling, were prepared for microscopic study by inflating the lungs with formalin at a pressure of 10 cm water. Paraffin sections were prepared and stained with hematoxylin and eosin. Replicate sections were stained for iron by the potassium ferrocyanide method and counterstained with van Gieson's stain. The topographic relationship of silica was demonstrated by the author's recently published photographic method.

Results

Rats. Initially, the iron was very closely associated with the silica, although negligible amounts of iron and silica were found to be independent of one another. Three months after injection of the iron-contaminated silica, the lungs contained scattered cellular aggregates or small nodules which showed no indication of collagen formation. These were apparently attached to alveolar walls or situated about vessels. They contained a scanty amount of iron as gauged by the diffuse green coloration of the nodules. The intensity of the color varied from one nodule to the next and was not necessarily uniform throughout the same nodule. The silica content of the nodules, on the other hand, was high regardless of the low iron content. This finding is, in effect, evidence of the dissociation of iron from the silica. Nearby air spaces contained scattered or clustered alveolar macrophages, most of which took a deep blue stain indicative of high iron content. Although a few siderotic macrophages appeared to be free of silica, most of them contained appreciable amounts of the latter.

In comparison, the control animals injected with iron-free silica showed nodules which at one month were as large as, or larger than, and as numerous as those of the iron-contaminated silica group at 3 or 4 months after the injection. Whereas at 6 months following injection, the nodules of control animals showed a moderate collagen content along with a fair cellularity, the nodules secondary to iron-contaminated silica were considerably smaller, more cellular, and contained little or no collagen.

The essential difference at this time between the animals injected with iron-free silica and those injected with iron-contaminated silica was a greater number of nodules and larger masses of confluent nodulations in the former.

Tracheal lymph nodes of the control animals showed collagenization which was moderate at 3 months and progressed rapidly thereafter.

Coincident with the collagenization there was an abundance of silica demonstrable in the lymphoid tissue. In the rats injected with iron-contaminated silica, collagenization of the lymph nodes was greatly delayed. At 3 to 4 months after injection, the lymph nodes contained small discrete endothelial foci but no collagen. The peripheral portions of these foci were rich in iron as well as in silica. At 11 to 16 months after injection, collagenization of the lymph nodes was well advanced, with some exceptions, however. The collagenized nodes contained abundant silica, but little or no iron. Where iron was present, it was associated with silica. The exceptions, where collagenization of the nodes was absent, contained little silica and little iron in spite of the presence of well defined pulmonary nodulations.

Guinea Pigs. Pulmonary response to inhalation of iron-contaminated silica dust was notably different than the intratracheal injection of the same dust in rats. The reaction in guinea pigs consisted of proliferation of alveolar cells which desquamated so as to fill many of the air spaces with cells or their debris, or both. This was associated with a non-collagenous thickening of alveolar walls, consisting of swelling and of proliferation of alveolar lining cells as well as interstitial edema. The entire lung was not uniformly affected. In the early state, there were widely separated, relatively small foci of this involvement. With continued exposure, the foci became larger and more numerous. The macrophages stained strongly for iron throughout the 12-month exposure period, but the iron content gradually diminished during the second year so that at the end of this time there were many foci which contained little or no iron. However, some demonstrable iron persisted in scattered foci. The silica content of the air spaces remained high irrespective of the diminishing iron content and regardless of whether the alveolar content consisted largely of well preserved cells or merely of cellular debris. Toward the end of the second year, scattered small silica dust aggregates were found within the thickened alveolar walls. This alveolar wall thickening was very pronounced and widespread at this time.

Cellular aggregates leading to formation of nodules never developed in these animals. With some exception, no collagen formation was demonstrable in the lungs even at the end of the second year. Exceptions were represented by several animals killed at the termination of the experiment in which a number of foci were found whose thickened alveolar walls contained coarse collagen fibers. Such alveolar fibrosis was not widespread; it was associated with abundant iron-free silica in adjoining air spaces. A puzzling observation was a similar abundance of iron-free silica elsewhere where no collagen was present.

Although foci of hyalinized collagen were found in the tracheal lymph nodes of some guinea pigs dead or killed 6 to 11 months following the last exposure to the same dust, iron was not demonstrable in either the nodes with fibrosis or those in which collagen was absent, but an abundance of silica was present in all nodes.

Summary

Inhibition of the fibrogenic action of quartz dust by associated iron, initially reported by Kettle and later confirmed by Gardner and Vorwald, has been reconfirmed.

In rats, this inhibition was temporary, serving to delay the maturation of silicotic nodules by a few months only. In guinea pigs, iron completely prevented the formation of silicotic nodules, but did not inhibit completely the development of collagen within thickened alveolar walls.

In rats, development of collagen within silicotic nodules paralleled the disappearance of iron from these sites. In guinea pigs, iron had also disappeared from those regions in which collagen had developed. Yet, there were other foci in which there was manifest inhibition of collagen formation in spite of the fact that abundant iron-free silica was demonstrable in such regions.

Apparent inhibition of fibrosis in the absence of iron contradicts Gardner's premise (applied to aluminum) that the inhibiting substance, to be effective, must occupy the same cell as the quartz particle which it inhibits. (P. Gross, M. L. Westrick, J. M. McNerney, *Experimental Silicosis: The Inhibitory Effect of Iron*: Dis Chest, 37: 35-41, January 1960)

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RESERVE



SECTION

Medical Department Inactive Duty Training

All Naval Reserve programs except surface, submarine, and aviation are special programs. Medical personnel are integrated in the air, surface, and submarine programs, and in all but a few of the special programs. They are also integrated into units of the Marine Corps Reserve.

All Naval Reserve programs except aviation are under the supervision of the Commander, Naval Reserve Training Command, Omaha, Neb., and the naval district commandants. Naval Air Reserve training is under the control of the Chief of Naval Air Reserve Training, Glenview, Ill. Medical personnel assigned to units of the Marine Corps Reserve are under the command of the Commandant of the Marine Corps.

Medical Department officers (MC, DC, MSC, & NC) who are members of the Ready Reserve are eligible for: (1) unit assignment with pay and without pay; (2) associate duty with pay and without pay; and (3) appropriate duty with

pay and without pay in the air, surface, and submarine programs, and in most of the special programs.

Medical Department officers who are members of the category Standby I of the Naval Reserve are excluded for participation in Selected Reserve activities, but may be members of units of special programs in a non-pay status.

Enlisted personnel of the Medical Department (hospital corpsmen and dental technicians) may be assigned with pay in units of the surface, submarine, and aviation programs with units of the Marine Corps Reserve, as members of Hospital Corps divisions, and as members of other special program units where a billet exists. They may be assigned without pay to Naval Reserve medical companies and to units of other special programs where a vacancy exists for an HM or DT rating.

There are many ways for Medical Department officers to participate in Naval Reserve training in a manner appropriate to their rank and designator, and thereby earn promotion and retirement points.

a. Medical officers may perform physical examinations; dental officers may perform dental examinations; and Nurse Corps officers may assist in these examinations of members of units of both the Naval Reserve and the Marine Corps Reserve.

b. Medical Corps and Medical Service Corps officers may be assigned as training officers with units of the surface program to provide training for hospital corpsmen assigned to such units.

c. Medical Department officers may be assigned, according to rank and designator, in the following capacities:

(1) As members of mobilization teams, medical companies, composite companies, and Naval Reserve Officers' Schools.

(2) As consultants at naval hospitals in accredited specialties.

(3) As Commandant's Representatives at medical schools.

Reserve Medical Department officers are strongly urged to become members of Medical Specialist Units, for these units comprise pools of reserve medical talent, and the commandants and commanding officers of USN and MCR Training Centers look to these units as sources of supply to fill vacant billets in their training programs. This is an element of support that must not be overlooked.

Furthermore, Reserve Medical Department officers are urged to take correspondence courses to insure earning sufficient retirement points to make continuous satisfactory years for retirement with pay and sufficient promotion points to enhance chances for promotion.

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On the Job Training

Medical Department Organization and Operations

On the Job Training in Medical Department organization and operations is available at any suitable training medical facility for Naval Reserve Medical Department personnel, male and female, who have had previous active duty for training. The convening date will be arranged between the commandant, trainee, and commanding officer of the training facility. This training is available in all naval districts with the exception of 10, 14, 15, and 17.

Submarine Medicine

A two-week course in On the Job Training in Submarine Medicine will convene on 7 November 1960 at the U.S. Naval Medical Research Laboratory, U.S. Naval Submarine Base, New London, Conn. This training will present an up-to-date review of problems relating to submarine medicine, including recent developments in submarine medicine research. Quotas for the course have been allocated to the 1st, 3rd, 9th, and 11th Naval Districts. Naval Reserve Medical Corps and Medical Service Corps, male officer personnel, of the above districts are eligible to request orders from their naval district commandant. SECRET clearance is required.

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Seminars for CO's of Reserve Medical Companies

Commanding officers, or their representatives, of Naval Reserve Medical companies will meet at either of two seminars to be conducted during November 1960. One meeting will be held in the Bureau of Medicine and Surgery, Washington, D. C., 7 - 9 November 1960; the other will be held at Headquarters, 9th Naval District, Great Lakes, Ill., 14 - 16 November 1960.

The seminars will provide training in the organization, administration, and operation of the Naval Reserve Program with particular emphasis on the medical components. Field trips to naval activities and other facilities will be conducted. A series of meetings will be held between the trainees and officers of the Bureau and/or district staffs with a view toward an improved Medical Reserve Program through the exchange of ideas and recommendations.

The 1st, 3rd, 4th, 5th, and 6th Naval Districts have been allocated quotas for the seminar convening in the Bureau of Medicine and Surgery; the 8th, 9th, 11th, 12th, and 13th Naval Districts have been allocated quotas for the seminar convening in the 9th District. Priority for orders should be given to commanding officers and executive officers in that order.

Annual Military Medico-Dental Symposium

The Eleventh Annual Military Medico-Dental Symposium will be held at the U.S. Naval Hospital, Philadelphia, Pa., 19 - 21 October 1960 under the sponsorship of the Commandant, Fourth Naval District.

The theme of this year's symposium will be "Recent Advances in Military Medicine." Subjects to be discussed will be under the following main topics: "Naval Space Medical Program," "Navy Medical Research Reports," "The Effect of the White House Conference on Our Children and Youth in the Coming Decade," and "Medical Appraisal of Functional Symptoms."

Inactive Naval Reserve officers may be credited with one retirement point credit for each day's attendance provided they register daily with the military representative present.

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DENTAL



SECTION

Effect of Heat on Cast Dental Gold Alloy

Metallographic investigation of a type IV, extra hard, dental gold alloy was undertaken to study the relationship between microstructure and hardness of a cast gold alloy which had been heat treated under various conditions.

The qualitative and quantitative composition of the gold used in this study was: gold, 71.1%; palladium 4.0%; silver, 9.0%; copper, 15.4%; and zinc, 0.5%.

Many interrelated factors must be considered in the successful casting of partial denture gold alloy which, if overlooked or carelessly handled, could adversely affect the desired characteristics of a casting. Subsequent heat treatments are designed to produce desired mechanical properties in a casting, but they cannot be expected to rectify the results of careless, irresponsible original work. Treatments consist of a "combination of heating and cooling operations, timed and applied to a metal or alloy in the solid state in a way that will produce desired properties."

Heat treatment widely used in dental annealing operations, and in this study, consists of placing the casting in a preheated furnace at 1290 F for

10 minutes and quenching in water at room temperature. (Annealing decreases hardness and strength and usually increases ductility). The microstructure is altered to a relatively homogeneous solid solution with a reduction of segregations. The precise mechanism is obscure, but it is believed to be brought about by means of some structural alteration by slow diffusion.

Hardening of gold alloys can be accomplished by one or two general methods, each of which has as its purpose the control of intermetallic transformations in solid solutions. (Hardening treatment increases hardness and strength and decreases ductility).

Results of Individual Heat Treatment Methods

Variable Time-Temperature Method. On the basis of observed findings, it appeared that hardness results of heat treating by quenching water, after the mold had been bench cooled for a given period of time, were of questionable value other than to obtain optimal conditions for subsequent heat treatment in order to modify existing segregations. A partial denture designed for casting is ordinarily of an irregular shape and of variable size and bulk. In addition, its positioning in the casting ring, and the factors of number, length, and manner of attachment of the sprues, all contribute to a nonuniform casting. Subsequent heat treatments are indicated to obtain greater uniformity.

Hardness results by the method of heat treatment involving annealing at 1290 F for 10 minutes and bench cooling is not recommended because of unpredictable results, dependent upon variable thermal environmental conditions.

Constant Time-Temperature Method. Those specimens, heat soaked in either a preheated furnace or a salt bath at 450 F for 5, 10, or 20 minutes, and subsequently quenched in water at room temperature, produced a microstructure similar to that found in an annealed condition, with an American Society Testing Materials (ASTM) grain size of No. 4.5. Specimens examined were found to be relatively free of segregations. This method of heat treating is preferred because of its simplicity and the uniformity of its results.

Polishing temperatures

To determine the amount of heat generated during the polishing of routine cast partial dentures, a copper vs. constant thermocouple was used in conjunction with a Leeds and Northrup potentiometer. The thermocouple was placed opposite the area in which the grinding was to be performed and made secure with adhesive tape. A minimum of six readings was made for each operation during the grinding and polishing procedures. The gauge

of the sprues was 0.06 inch. The dental lathe was operated at high speed (3400 rpm). Temperatures were recorded during the cutting and the subsequent reduction of the sprues and the polishing of the clasps. Findings indicated that the grinding and polishing of cast partial dentures elevated the local temperature of the material, but not beyond 200 F. The heating was of short duration, not great enough to transform the solid solution, and considered harmless. (CAPT A. K. Kaires DC USN, J. C. Thompson, Effect of Heat on Cast Dental Gold Alloy: J Dent Res, 38: 888-900, Sep - Oct 1960)

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Risk of Fire and Explosion in the Dental Office

Disregard of the elementary safety and precautionary measures by dentists, dental assistants, or patients may lead to serious accidents, such as fire or explosion. Safety regulations should be posted in every room of the dental office, clearly visible to the dentist's personnel and his patients.

All inflammable anesthetic preparations—which usually contain oxygen—are essentially explosive. Almost as dangerous are chemical disinfectants, such as benzene, alcohol, and their derivatives.

Smoking in the dental office should be prohibited. Static electricity which may ignite explosive compounds is a fire and explosion hazard which exists in many operating rooms. Clothing made of silk, wool, nylon, or other synthetic textures exhibits a maximum sparking potential.

Anesthesia apparatus as well as all parts of the electric equipment should be properly insulated and periodically checked. Defects in electric wiring may cause fire and explosion in the operating room.

Containers of inflammable anesthetics should be painted in different colors, such as nitrous oxide in light blue, cyclopropane in orange, ethylene in red, and oxygen in green.

Occasionally, the physical condition of a patient or the requirements of a specific dental or oral surgical procedure may offer temptation to violate some of the safety regulations. The fact that 36 explosions occurred in dental offices in England between 1947 and 1954 illustrates the possible result of such violations. The report of the British Ministry of Health on explosions in the operating theater (1956) demonstrated that had all precautionary measures been followed, these accidents would not have occurred. (F. Duyvenesz: Tschr. tandheelk. 65: 113-124, February 1959)

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Personnel and Professional Notes

CAPT Pollard Selected for Rear Admiral. CAPT Eric G. F. Pollard DC USN, Commanding Officer, U. S. Naval Dental School, Bethesda, Md., has been

selected for promotion to the rank of Rear Admiral. CAPT Pollard was born in Cleveland, Ohio, and matriculated at the University of Minnesota from which he received his D.D.S. in 1933. Among many assignments ashore and afloat during more than 24 years of active duty in the Navy Dental Corps, a memorable tour was with the U.S. Embassy Guard, North China Marines, Peking, China, where he served concurrently as Honorary Lecturer in Dentistry and Acting Head of the Dental Service at Peiping Union Medical College.

This association was abruptly terminated by the outbreak of World War II and the detention of the U. S. Embassy Guard by components of the Imperial Japanese Army. Although a prisoner of war for the duration of the war, CAPT Pollard was able to render dental treatment to fellow prisoners and was the anesthetist and assistant surgeon of the POW medical team. For his service and accomplishments while a prisoner, he was awarded the Legion of Merit and Purple Heart. CAPT Pollard is a Fellow of the American College of Dentists, and a member of the American Academy of Oral Pathology, Omicron Kappa Upsilon, Sigma Alpha Epsilon, and the American Dental Association.

Dental Reserve Seminar. The Seminar for Commanding Officers of Dental Reserve companies has been rescheduled for 30 October 1960 vice 6 November 1960 as outlined in BuPers Instruction 1571.4F. This change was considered desirable as it makes the seminar coincide with the Meeting of Military Surgeons and enables the conferees to attend some of the outstanding programs that will be presented at that time.

Inspector Dental Activities, Atlantic Coast. The office of the Inspector Naval Dental Activities, Atlantic Coast has been disestablished by SecNav Notice 5450.

New Commanding Officers. In consonance with the policy of affording desired training to as many officers as possible, the following changes in commanding officers of Naval Reserve Dental companies in the 12th Naval District have been effected:

NRDC 12-2 Cdr John P. Bradley for CAPT Bill J. Harris
NRDC 12-5 CDR William S. Wilson for CDR George N. Fitzgerald
NRDC 12-7 LCDR John F. Bihn for CDR John L. Richards

LCDR Ricker Relieves CDR Jacobs. LCDR R. E. Ricker MSC USN has relieved CDR J. J. Jacobs MSC USN as Head, Enlisted Personnel Section, Dental Division, Bureau of Medicine and Surgery. LCDR Ricker was last assigned as Executive Assistant to the Inspector General, Dental. CDR Jacobs will attend the School of Naval Justice in Newport, R. I., prior to reporting to his new assignment as Administrative Assistant to the Commanding Officer, U. S. Naval Dental Clinic, Naval Weapons Plant, Washington, D. C.

Vacuum Fired Porcelain Furnace. Efforts toward improving the processing of dental porcelain through use of a vacuum fired porcelain furnace have been accomplished at the dental department, Mare Island Naval Shipyard. The furnace, utilizing a surveyed vacuotrol unit and a S.S. White Electric Furnace No. 3, was constructed by personnel of the Shipyard public works and dental department. CAPT M. J. Crawford DC USN is Senior Dental Officer at the Shipyard.

Dr. Brecker Lectures at NDS. Dental practitioner, clinician, teacher, author, and staff member of Columbia University, Dr. S. Charles Brecker was a recent guest in the special lectures series presented by the U. S. Naval Dental School to Dental officers of the Armed Forces, civilian dentists, and other scientific personnel of the Washington, D. C. area. The illustrated lecture covered difficulties encountered in mouth restoration; discussed factors that must be considered in treating patients with periodontal disease, occlusal disharmonies, and oral rehabilitation problems; and stressed the need for adequate diagnosis and conservatism both in tooth preparation and in restorative procedures. At a conference after the lecture, examples of porcelain fused to various metals were shown, and problems of esthetics, mechanical strength, construction, fracture, and repair were discussed. Dr. Brecker also illustrated designs for abutments and pontics where porcelain fused to metal might be used to good advantage.

RADM Schantz to Serve on Commission. RADM C. W. Schantz DC USN, Assistant Chief of the Bureau of Medicine and Surgery (Dentistry) and Chief, Dental Division was elected to serve for 3 years as a member of the F. D. I. commission on Armed Forces Dental Services.

CAPT McKee Retires. CAPT D. L. McKee DC USN was transferred to the retired list of the Navy on 1 August 1960 after more than 20 years' service. CAPT McKee was born in Cherokee, Iowa, and in 1921 graduated from the Dental School, Northwestern University, Chicago, Ill. He conducted a private practice of dentistry in Vermillion, S. D., and in Sioux City, Iowa. From 1926 through 1936, he held a commission in the Army Dental Corps Reserve. In May 1942, CAPT McKee accepted a commission as Lieutenant Commander in the Navy Dental Corps Reserve and reported for duty to the Naval Training Center, Great Lakes, Ill. In July 1946, he transferred to the Regular Navy.

CAPT McKee served at many duty stations, among which was Fleet Hospital #117 and Naval Operating Base, Okinawa. He also served as Staff Dental Officer for the Chief, Naval Air Reserve Training and for the Chief of Naval Air Training. Prior to his retirement, CAPT McKee was District Dental Officer of the Sixth Naval District.

Study Shows Fluorides Safe. There is no evidence that fluorides in water have a harmful effect on kidney structure or function. This is the finding of a recent study conducted by Dr. Harold C. Hodge, pharmacology professor at the University of Rochester School of Medicine and Dentistry. The report by Dr. Hodge was made before a symposium held in Boston and sponsored by the AMA's Council on Foods and Nutrition. The Professor said that fluorides are quickly removed from body fluids and excreted rapidly and almost entirely in the urine. (ADA News Letter, June 1960)

Graduation of Dental Officers. Thirty-seven Dental Ensigns (1925) graduated from the U. S. Naval Officers' Candidate School, Naval Base, Newport, R. I., 21 July 1960. This was the first of five classes to be convened during this summer. CAPT H. J. Wunderlich DC USNR, Head, Reserve Branch, Dental Division, BuMed, addressed the graduates and extended the congratulations of RADM Schantz. Presentation of certificates was made by CAPT W. H. Snyder DC USN, and CAPT R. F. Erdman DC USN. ENS W. J. Kelly USNR, of Allentown, Pa., was named as Honorman and was presented a plaque.

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